

## SCREENING-LEVEL HAZARD CHARACTERIZATION

### **2-Nitropropane (CASRN 79-46-9)**

The High Production Volume (HPV) Challenge Program<sup>1</sup> was conceived as a voluntary initiative aimed at developing and making publicly available screening-level health and environmental effects information on chemicals manufactured in or imported into the United States in quantities greater than one million pounds per year. In the Challenge Program, producers and importers of HPV chemicals voluntarily sponsored chemicals; sponsorship entailed the identification and initial assessment of the adequacy of existing toxicity data/information, conducting new testing if adequate data did not exist, and making both new and existing data and information available to the public. Each complete data submission contains data on 18 internationally agreed to “SIDS” (Screening Information Data Set<sup>1,2</sup>) endpoints that are screening-level indicators of potential hazards (toxicity) for humans or the environment.

The Environmental Protection Agency’s Office of Pollution Prevention and Toxics (OPPT) is evaluating the data submitted in the HPV Challenge Program on approximately 1400 sponsored chemicals by developing hazard characterizations (HCs). These HCs consist of an evaluation of the quality and completeness of the data set provided in the Challenge Program submissions. They are not intended to be definitive statements regarding the possibility of unreasonable risk of injury to health or the environment.

The evaluation is performed according to established EPA guidance<sup>2,3</sup> and is based primarily on hazard data provided by sponsors; however, in preparing the hazard characterization, EPA considered its own comments and public comments on the original submission as well as the sponsor’s responses to comments and revisions made to the submission. In order to determine whether any new hazard information was developed since the time of the HPV submission, a search of the following databases was made from one year prior to the date of the HPV Challenge submission to the present: (ChemID to locate available data sources including Medline/PubMed, Toxline, HSDB, IRIS, NTP, ATSDR, IARC, EXTOXNET, EPA SRS, etc.), STN/CAS online databases (Registry file for locators, ChemAbs for toxicology data, RTECS, Merck, etc.) and Science Direct. OPPT’s focus on these specific sources is based on their being of high quality, highly relevant to hazard characterization, and publicly available.

OPPT does not develop HCs for those HPV chemicals which have already been assessed internationally through the HPV program of the Organization for Economic Cooperation and Development (OECD) and for which Screening Initial Data Set (SIDS) Initial Assessment Reports (SIAR) and SIDS Initial Assessment Profiles (SIAP) are available. These documents are presented in an international forum that involves review and endorsement by governmental authorities around the world. OPPT is an active participant in these meetings and accepts these documents as reliable screening-level hazard assessments.

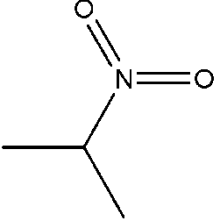
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<sup>1</sup> U.S. EPA. High Production Volume (HPV) Challenge Program; <http://www.epa.gov/chemrtk/index.htm>.

<sup>2</sup> U.S. EPA. HPV Challenge Program – Information Sources; <http://www.epa.gov/chemrtk/pubs/general/guidocs.htm>.

<sup>3</sup> U.S. EPA. Risk Assessment Guidelines; <http://cfpub.epa.gov/ncea/raf/rafguid.cfm>.

These hazard characterizations are technical documents intended to inform subsequent decisions and actions by OPPT. Accordingly, the documents are not written with the goal of informing the general public. However, they do provide a vehicle for public access to a concise assessment of the raw technical data on HPV chemicals and provide information previously not readily available to the public.

<p><b>Chemical Abstract Service Registry Number (CASRN)</b></p>	<p><b>79-46-9</b></p>
<p><b>Chemical Abstract Index Name</b></p>	<p><b>Propane, 2-nitro-</b></p>
<p><b>Structural Formula</b></p>	
<p style="text-align: center;"><b>Summary</b></p> <p>CASRN 79-46-9 is a colorless liquid possessing high vapor pressure and high water solubility. It is expected to possess high mobility in soil. Volatilization is considered moderate based on its Henry's Law constant. The rate of hydrolysis is negligible. The rate of atmospheric photooxidation is also negligible. CASRN 79-46-9 is expected to have low (P1) to moderate (P2) persistence and low bioaccumulation potential (B1).</p> <p>The acute oral and dermal toxicity of CASRN 79-46-9 is low in rabbits. The acute inhalation toxicity of CASRN 79-46-9 is high in rats. In the 6-month repeated-dose inhalation toxicity study in rats, CASRN 79-46-9 showed increased liver weights, increased serum glutamic-pyruvic transaminase (SGPT) activity and liver histopathology and liver basophilic foci with hepatocyte hyperplasia and hyperchromatic nuclei at 0.8 mg/L/day; the NOAEC for systemic toxicity is 0.1 mg/L/day. In an 18-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed decreased body weight, increased liver weights, liver lesions at necropsy, increased SGPT, liver histopathology including focal necrosis, vascular degeneration and nodular hyperplasia and increased renal calcification at 0.4 mg/L (lowest concentration tested); the NOAEC was not established. In a 22-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed increased absolute and relative liver weight, focal vacuolization of hepatocyte cytoplasm and kidney calcification at 0.09 mg/L/day (lowest concentration tested); the NOAEC was not established. In a 6-month inhalation repeated-dose toxicity study in rabbits, CASRN 79-46-9 showed congestion and hemorrhage of the lungs with interstitial edema and necrosis in the hemorrhagic areas at 0.8 mg/L after 28-days; the NOAEC for systemic toxicity is 0.1 mg/L/day. No specific reproductive toxicity studies are available. However, in the 22-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed no treatment-related effects on the reproductive organs (prostate, seminal vesicles, testis, uterus and ovaries). In the inhalation dominant lethal study in rats, CASRN 79-46-9 showed no treatment-related effects. CASRN 79-46-9 induced gene mutations in bacteria <i>in vitro</i> and chromosomal aberrations in mammalian cells <i>in vivo</i>. CASRN 79-46-9 did not induce chromosomal aberrations or dominant lethal mutations in mammalian cells <i>in vitro</i> and <i>in vivo</i>, respectively. CASRN 79-46-9 is irritating to rabbit eyes, not irritating to rabbit skin and not a skin sensitizer in guinea pigs. CASRN 79-46-9 increased the incidence of liver and lung tumors in rats via the</p>	

oral route. CASRN 79-46-9 increased the incidence of tumors in rats exposed via the inhalation route but not in rabbits.

The 96-h LC<sub>50</sub> value of CASRN 79-46-9 for fish is >210 mg/L. The 48-h EC<sub>50</sub> value of CASRN 79-46-9 for aquatic invertebrates is 19 mg/L. The 72-h EC<sub>50</sub> values of CASRN 79-46-9 for aquatic plants are 267 and > 887 mg/L for biomass and growth rate, respectively.

The reproductive/developmental toxicity endpoints were identified as data gaps under the HPV Challenge program.

The sponsor, The Dow Chemical Company, submitted a Test Plan and Robust Summaries to EPA for 2-nitropropane (CASRN 79-46-9; 9<sup>th</sup> CI name: propane, 2-nitro-) on May 18, 2005. EPA posted the submission on the HPV Challenge website on June 3, 2005 (<http://www.epa.gov/chemrtk/pubs/summaries/2nitropne/c15898tc.htm>). EPA comments on the original submission were posted to the website on March 7, 2007. Public comments were also received and posted to the website. The sponsor submitted updated/revised documents on May 16, 2007, which were posted to the website on August 20, 2007.

## **Justification for Supporting Chemical**

In the revised test plan, the sponsor proposed the use of data for 1-nitropropane (CASRN 108-03-2) to address the reproductive/developmental toxicity endpoint for human health. Studies with rat liver enzymes (Haas-Jobelius et al., 1992) suggest fundamental differences in metabolism between 1-nitropropane and 2-nitropropane. This is supported by differences in data for genotoxicity and carcinogenicity. Therefore, EPA does not consider data for CASRN 108-03-2 appropriate to address the human health endpoints for CASRN 79-46-9 for the purposes of the HPV Challenge program.

### **1. Chemical Identity**

#### **1.1 Identification and Purity**

CASRN 79-46-9 is a colorless liquid with a molecular formula of  $\text{CH}_3\text{CH}(\text{NO}_2)\text{CH}_3$  and a typical purity of the commercial material is  $\geq 94\%$ .

#### **1.2 Physical Chemical Properties**

Propane, 2-nitro- is a colorless liquid possessing high vapor pressure and high water solubility. The physical-chemical properties of propane, 2-nitro are summarized in Table 1.

<b>Table 1. Physical-Chemical Properties of Propane, 2-nitro-<sup>1</sup></b>	
<b>Property</b>	<b>Value</b>
CASRN	79-46-9
Molecular Weight	89.09
Physical State	Colorless liquid
Melting Point	-91.3 °C (measured); -93 °C (measured)
Boiling Point	120.2 °C (measured)
Vapor Pressure	13.0 mm Hg at 20°C (measured); 17.3-20.1 mm Hg at 25°C (measured)
Water Solubility	17,000 –17,400 mg/L at 25°C (measured)
Dissociation Constant (pK <sub>a</sub> )	Not applicable
Henry's Law Constant	1.2×10 <sup>-4</sup> atm-m <sup>3</sup> /mol (estimated) <sup>2</sup>
Log K <sub>ow</sub>	1.35 (measured)

<sup>1</sup> Dow Chemical Company. May 16, 2007. Revised Test Plan and Robust Summary for 2-Nitropropane. Available online from: <http://www.epa.gov/chemrtk/pubs/summaries/2nitropne/c15898tc.htm> as of November 1, 2010.

<sup>2</sup> U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online from: <http://www.epa.gov/opptintr/exposure/pubs/episuitedl.htm> as of November 2, 2010.

## **2. General Information on Exposure**

### **2.1 Production Volume and Use Pattern**

According to the 2006 IUR submissions, CASRN 79-46-9 had an aggregated production and/or import volume in the United States between 50 and 100 million pounds.

Industrial processing and uses are claimed confidential. No commercial and consumer uses were reported for this chemical.

### **2.2 Environmental Exposure and Fate**

Propane, 2-nitro- is expected to possess high mobility in soil. Propane, 2-nitro- present at 2.0 and 9.9 mg/L, reached 14 and 8% of its theoretical biochemical oxygen demand (BOD), respectively in 4 weeks using an activated sludge inoculum at 2 mg/L and the MITI-I (OECD 301C) test. It was also not readily biodegradable in a separate closed bottle (OECD 301D) test, achieving 0.1% of its theoretical BOD after a 28 day incubation period at an initial concentration of 2 mg/L. The biodegradation rate of propane, 2-nitro- was studied under laboratory conditions using an acidic (pH 4.8) clay soil with <1% organic matter and a slightly alkaline (pH 7.8) sandy loam containing 3.25% organic matter. The half-life of propane, 2-nitro- was 0.66 days in the acidic soil and 0.5 days in the alkaline soil; however, no abiotic controls were employed in these studies and it was possible some of the loss was due to volatilization. Aerobic biodegradation studies performed with soil/water suspensions resulted in 3.0% conversion to CO<sub>2</sub> in 35 days. During this time 28.8% was lost as volatile products. The rate of hydrolysis of propane, 2-nitro- is negligible. The rate of volatilization is considered moderate based on its Henry's Law

constant. Propane, 2-nitro- is expected to have low (P1) to moderate persistence (P2) and low bioaccumulation potential (B1).

The environmental fate data are provided in Table 2.

<b>Property</b>	<b>Value</b>
Photodegradation Half-life	63.4 days (estimated) <sup>2</sup>
Hydrolysis Half-life	Stable
Biodegradation	0.1% after 28 days( not readily biodegradable) 8-14% after 28 days (not readily biodegradable) <sup>3</sup> ; Half-life of 0.66 days in an acidic clay soil and 0.5 days in an alkaline sandy loam <sup>4</sup> ; 3% degradation after 35 days using soil/water suspensions <sup>5</sup>
Bioaccumulation Factor	BCF = 1 (measured in golden orfe at 0.050 mg/L); BCF = 0.9 –1.1 (measured in carp at 2 mg/L) <sup>3</sup> ; BCF = <8.4 (measured in carp at 0.20 mg/L) <sup>3</sup> BAF = 1.5 (estimated) <sup>2</sup>
Log K <sub>oc</sub>	1.5 (estimated) <sup>2</sup>
Fugacity (Level III Model) <sup>2</sup>	
Air (%)	13.5
Water (%)	30.4
Soil (%)	56.0
Sediment (%)	0.1
Persistence <sup>6</sup>	P1(low) – P2 (moderate)
Bioaccumulation <sup>6</sup>	B1 (low)

<sup>1</sup> Dow Chemical Company. May 16, 2007. Revised Test Plan and Robust Summary for 2-Nitropropane. Available online from: <http://www.epa.gov/chemrtk/pubs/summaries/2nitropne/c15898tc.htm> as of November 1, 2010.

<sup>2</sup> U.S. EPA. 2010. Estimation Programs Interface Suite™ for Microsoft® Windows, v4.00. U.S. Environmental Protection Agency, Washington, DC, USA. Available online from: <http://www.epa.gov/opptintr/exposure/pubs/episuitedi.htm> as of November 2, 2010.

<sup>3</sup> National Institute of Technology and Evaluation. 2002. Biodegradation and Bioaccumulation of the Existing Chemical Substances under the Chemical Substances Control Law. Available online from: [http://www.safe.nite.go.jp/english/kizon/KIZON\\_start\\_hazkizon.html](http://www.safe.nite.go.jp/english/kizon/KIZON_start_hazkizon.html) as of November 2, 2010.

<sup>4</sup> Loehr RC, Matthews JE. 1992. J. Soil Communication 1(4): 339-360.

<sup>5</sup> Freitag D., Korte S., Korte F. 1988. Ecotoxicological profile analysis of nitroparaffins according to OECD guidelines with C14-labelled compounds. In: TSCA SECT. 8D Submiss. To EPA for nitromethane (fiche no. OTS516767 Doc. No. 86-890000232) and nitroethane (fiche no. OTS51678 Doc. No. 86-890000233)

<sup>6</sup> Federal Register. 1999. Category for Persistent, Bioaccumulative, and Toxic New Chemical Substances. *Federal Register* 64, Number 213 (November 4, 1999) pp. 60194–60204.

**Conclusion:** Propane, 2-nitro- is a colorless liquid possessing high vapor pressure and high water solubility. It is expected to possess high mobility in soil. Volatilization is considered moderate based on its Henry’s Law constant. The rate of hydrolysis is negligible. The rate of atmospheric photooxidation is also negligible. Propane, 2-nitro- is expected to have low (P1) to moderate (P2) persistence and low bioaccumulation potential (B1).

### 3. Human Health Hazard

A summary of the human health toxicity data submitted for SIDS endpoints is provided in Table 3. CASRN 79-46-9 was assessed in the OECD HPV Program as a Targeted Assessment (SIAM 30; <http://webnet.oecd.org/hpv/ui/Search.aspx>) which is not considered a full SIDS assessment. The endpoints addressed were genotoxicity and carcinogenicity.

#### *Acute Oral Toxicity*

Rabbits (strain, sex and number not specified) were administered 2-nitropropane via gavage (dose level and length of observation period not specified). Observations noted 20 – 40 minutes following dose administration included progressive weakness and collapse, unsteadiness, loss of coordination ending in ataxia and changes in respiration patterns. No hematological effects were noted.

**LD<sub>50</sub> = 500 – 750 mg/kg**

#### *Acute Inhalation Toxicity*

(1) Sprague-Dawley rats (8 males/concentration; 10 females/concentration; 8/sex/control group) were exposed whole-body to continuously-monitored concentrations of 2-nitropropane as a vapor at 0 (water vapor control), 367, 405, 461 or 574 ppm (~ 1.3, 1.5, 1.7 or 2.1 mg/L) (males) and 0 (water vapor control), 370, 416, 464, 602 or 805 ppm (~ 1.3, 1.5, 1.7, 2.2 or 2.9 mg/L) (females) for 6 hours and observed for 14 days. Mortality occurred at  $\geq 405$  ppm in males and at 805 ppm in females. Weight gain was not affected in males; however, the surviving females at 805 ppm did not gain weight throughout the duration of the study. Both male and female rats moved about the chamber immediately upon exposure, followed by slight depression and hyperventilation. Cyanosis was observed at concentrations of  $\geq 405$  ppm in males and  $\geq 602$  ppm in females. Necropsies revealed no treatment-related findings.

**LC<sub>50</sub> (males) ~ 1.5 mg/L**

**LC<sub>50</sub> (females) ~ 2.6 mg/L**

(2) Rats (strain and sex not specified) were exposed to 2-nitropropane (inhalation route not specified) at 80, 760 or 14,700 ppm (~ 0.3, 2.8 or 53.6 mg/L). Five animals were exposed at the lowest concentration 8 hours/day for 5 days, four animals were exposed at the mid-concentration 8 hours/day for 1 or 2 days and one animal was exposed at the highest concentration for 4 hours. Methemoglobin (MetHb) concentrations in blood and 2-nitropropane concentrations in the liver, lung, heart and kidney were measured. The animal exposed to the highest concentration died within 4 hours of exposure. One animal exposed to the mid-concentration level for 1 day survived the exposure period and was euthanized. The other mid-concentration animal that was exposed for 1 day was allowed to recover and died 48 hours following exposure. The two mid-concentration animals from the 2-day exposure group died 2 hours after the last exposure. All low-concentration animals survived. In the high-concentration animal, the blood MetHb concentration was 84% and 2-nitropropane (23 mg/100 g) was found in the liver. 2-Nitropropane (18 mg/100 g) was also found in the liver of mid-concentration animals (exposure

duration not specified). In low-concentration animals, no methemoglobin was found in blood and no 2-nitropropane was found in the liver.

**LC<sub>100</sub> = ~ 53.6 mg/L**

(3) ICR mice (number/sex/concentration not specified) were exposed whole-body to continuously-monitored concentrations of 2-nitropropane as a vapor at 0 (water vapor control), 454, 558 or 738 ppm (~ 0, 1.7, 2.0 or 2.7 mg/L) (males) and 0 (water vapor control), 495, 640 or 740 ppm (~ 0, 1.8, 2.3 or 2.7 mg/L) (females) for 6 hours and observed for 14 days. Seven males from the mid-concentration group died within 14 days and nine males from the high-concentration group died within 7 days. Two low-concentration females and 11 mid-concentration females died within 7 days and all 14 high-concentration females died within 14 days. There were no effects on body weight gain. Both male and female rats moved about the chamber immediately upon exposure, followed by slight depression (both sexes) and hyperventilation (males). Cyanosis was observed in males at 558 and 738 ppm and in females at 640 ppm and 740 ppm. Necropsies revealed no remarkable findings; however, some petechial hemorrhage of the lung was noted in males.

**LC<sub>50</sub> ~ 2.0 mg/L**

### ***Acute Dermal Toxicity***

Ten New Zealand White rabbits (distribution by sex not specified) were administered 2-nitropropane (4.4 – 5.7 mL; 97.10% purity; containing 1.01% nitroethane, 2.59% 1-nitropropane and 0.023% water by weight) via the dermal route (abraded skin) at 2000 mg/kg-bw under occluded conditions for 24 hours and observed for 14 days. No mortality, signs of toxicity, erythema or edema were observed and no effects on body weight gain were noted.

**LD<sub>50</sub> > 2000 mg/kg**

### ***Repeated-Dose Toxicity***

(1) Male Sprague-Dawley rats (10/group) were exposed whole-body to 2-nitropropane (94.45% pure; containing 3.07% 1-nitropropane, 1.96% nitroethane, 0.42% 2-nitro-2-methylpropane, 0.03% 2-nitrobutane and < 0.01% water by weight) as a vapor at 27 or 207 ppm (~ 0.1 or 0.8 mg/L) 7 hours/day, 5 days/week for 2 or 10 days or 1, 3 or 6 months. There were no treatment-related effects on body weight. Changes were noted in some hematologic endpoints (hematocrit, hemoglobin, red blood cells, prothrombin time and MetHb), but the changes were not clearly treatment-related because of inconsistent direction of change or absence of concentration-response or exposure duration-response relationships. At the high concentration, serum glutamic-pyruvic transaminase (SGPT) was increased in animals exposed for 10 days, 1 month and 6 months. An increase in serum thyroxin was not clearly treatment-related because the increase occurred only in high-concentration animals treated for 3 months. At 1, 3 and 6 months, high-concentration animals exhibited increased incidences of dark hemorrhagic foci in the lungs at necropsy. High-concentration animals from the 1-, 3- and 6-month exposure group and low-concentration animals from the 3-month exposure group exhibited increased wet/dry lung weights, and high-concentration animals showed dark hemorrhagic foci at necropsy.

Histopathology of lungs at necropsy was unremarkable. The livers of high-concentration animals exposed for 3 months had areas of necrosis and surface lesions and were pale in color. Relative liver weights of high-concentration animals were also increased at 3 and 6 months. After 6 months of exposure, the livers of high-concentration animals were enlarged and pale with numerous masses and lesions. Multiple focal areas of hepatocellular hypertrophy were noted in 9/9 high-concentration animals from the 3-month exposure group. The hepatocytes in these foci were more eosinophilic than the surrounding cells and usually contained large, vesiculated nuclei. In four of the nine rats at the high concentration, basophilic foci containing hyperplastic, small hepatocytes with hyperchromatic nuclei were observed at 3 months. Occasionally, mitotic figures were present. Liver neoplasms were seen in the high-concentration group at 6 months (see carcinogenicity section below). There were no gross pathological or histopathological findings in any organ at the low concentration.

**LOAEC ~ 0.8 mg/L/day** (based on increased liver weights, increased serum SGPT and liver histopathology including hepatocyte hypertrophy and liver basophilic foci with hepatocyte hyperplasia and hyperchromatic nuclei)

**NOAEC ~ 0.1mg/L/day**

(2) Sprague-Dawley rats (125/sex/concentration) were exposed by whole-body inhalation to 2-nitropropane (95.65% pure; contains 3.63% 1-nitropropane, 0.20% nitroethane, 0.51% 2-nitro-2-methylpropane and 0.01% water by weight) as a vapor at 0 (control) or 100 ppm (~ 0.4 mg/L) 7 hours/day, 5 days/week for up to 18 months. Ten animals/sex/group were euthanized at 1-, 3-, 6-, 9- or 12-month intervals. Control animals (62 males and 67 females) and exposed animals (23 males and 30 females) were euthanized at 18 months. Additional groups were exposed to 2-nitropropane for 3 months (7 males and 10 females), 6 months (8 males and 10 females) and 9 months (7 males and 8 females) and euthanized at 18 months. Mortalities were not specified. Males from the 18-month exposure group exhibited decreased body weights and increased SGPT. Increased absolute and relative liver weights and hepatic lesions were observed in males exposed for 9, 12 or 18 months, with the frequency of hepatic lesions increasing with length of exposure (6/16, 5/10 and 22/23, respectively). Hepatic lesions were also found in the males that were found dead or moribund (11/16) and in females exposed for 9 and 18 months (1/20 and 2/30, respectively). No treatment-related effects on hematology or any other clinical chemistry parameter were noted in main group animals. Effects noted in recovery groups included decreased body weights in males exposed for 6 and 9 months and increased SGPT (compared to males exposed for 18 months with no recovery period), serum ornithine carbamyl transferase and MetHb in males exposed for 9 months. Recovery males exposed for 3, 6, or 9 months exhibited increased absolute and relative liver weights as well as hepatic lesions (6/10, 7/10 and 7/10, respectively). Lesions were also found in the livers of recovery females exposed for 9 months (2/10). The non-neoplastic liver lesions found in exposed males and females included focal necrosis, vacuolar degeneration and nodular hyperplasia (designation of effects to main study exposure groups and/or recovery groups not specified). There was also an increase in renal calcification in exposed males and females. Findings for neoplasms are described in the carcinogenicity section below.

**LOAEC ~ 0.4 mg/L/day** (based on decreased body weight, increased liver weights, liver lesions at necropsy, increased SGPT, liver histopathology including focal necrosis, vacuolar degeneration and nodular hyperplasia and increased renal calcification)

**NOAEC = Not established**

(3) Sprague-Dawley rats (125/sex/concentration) were exposed by whole-body inhalation to 2-nitropropane (95.65% pure; contains 3.63% 1-nitropropane, 0.20% nitroethane, 0.51% 2-nitro-2-methylpropane and 0.01% water by weight) as a vapor at 0 (control) or 25 ppm (~ 0.09 mg/L) 7 hours/day, 5 days/week for up to 12 months. Ten animals/sex/concentration were euthanized at 1-, 3-, 9- or 12-month intervals. Ten animals/group were also euthanized at 6 months with the exception of exposed males (N = 9). Sixty-two and 44 control males and females and 27 and 29 exposed males and females (respectively) were euthanized at 22 months. Additional recovery groups were exposed for 3 months (6 males and 8 females) or 12 months (7 males and 9 females) and euthanized at 22 months. Body and absolute liver weights of females exposed for 6, 12 or 22 months were increased. Both relative and absolute liver weights of males exposed for 22 months were increased and relative liver weights of males exposed for 6 months were increased. Absolute kidney weights of females exposed for 1 month and males exposed for 12 months were increased (relative kidney weight data were not presented). Ornithine carbamyl transferase activity was increased in males from the 12-month exposure group and decreased in males from the 3- or 6-month exposure groups. Females from the 12-month exposure group exhibited a decrease in serum thyroxin. Hemoglobin and hematocrit were increased in males from the 12-month exposure group and hematocrit was increased in females from the 6- or 12-month groups. Erythrocyte and white blood cell counts were increased in males exposed for 6 months and erythrocyte counts were increased in females exposed for 12 months. Recovery females exposed for 3 months exhibited increased body weights. Serum thyroxin was increased in recovery females from the 3- and 12-month exposure groups and MetHb was increased in recovery females from the 12-month exposure group. Erythrocyte counts were increased in recovery males exposed for 3 months. White blood cells were increased in recovery females exposed for 3 or 12 months. One liver angioma was observed in a control animal at 22 months and one liver adenoma was observed in an exposed female that was moribund after 21.5 months. The hepatocytes in the adenoma were uniform and had normal appearing nuclei. Focal vacuolization of the cytoplasm of hepatocytes were observed in control males, exposed males, control females and exposed females (22/125, 58/125, 18/125 and 19/124, respectively). The cells in the nodule areas were generally hypertrophied, but nuclei were normal. Calcification of the kidney was noted in 76 control animals and 94 exposed animals. Changes in body weight, serum chemistry and hematology were not considered to be treatment-related due to variations in control animals and no relationship of the changes to duration of exposure.

**LOAEC ~ 0.09 mg/L/day** (based on increased absolute and relative liver weight, focal vacuolization of hepatocyte cytoplasm and kidney calcification)

**NOAEC = Not established**

(4) New Zealand White rabbits (5/group) were exposed whole-body to 2-nitropropane (94.45% pure; containing 3.07% 1-nitropropane, 1.96% nitroethane, 0.42% 2-nitro-2-methylpropane, 0.03% 2-nitrobutane and < 0.01% water by weight) as a vapor at 27 or 207 ppm (~ 0.1 or 0.8 mg/L) 7 hours/day, 5 days/week for 2 days, 10 days, 1 month, 3 months or 6 months. There were no treatment-related effects on body weight at either concentration. Effects noted in low-concentration animals included an increase in ornithine carbamyl transferase activity at 3 months, which appeared to be due to one very high value. At 6 months, ornithine carbamyl transferase activity was decreased. Effects at the high concentration included a decrease in prothrombin time and elevated ornithine carbamyl transferase levels at 1 and 3 months. There

were no signs of liver or brain edema and no treatment-related effects on absolute or relative organ weights or gross pathology at either concentration. Three of the five high-concentration rabbits exposed for 1 month exhibited focal areas of moderate to moderately severe hemorrhage and congestion of the alveolar and alveolar duct walls. Interstitial edema and necrosis were present in the hemorrhagic areas. Histopathology of the lungs and other organs was normal in rabbits exposed for 3 or 6 months.

**LOAEC (28-day) ~ 0.8 mg/L** (based on congestion and hemorrhage of the lungs with interstitial edema and necrosis in the hemorrhagic areas)

**NOAEC ~ 0.1 mg/L**

**NOAEC (6 month) ~ 0.8 mg/L** (highest concentration tested)

### ***Reproductive Toxicity***

No reproductive toxicity data are available. However, in the 22-month inhalation repeated-dose toxicity study in rats described above, gross pathological and histopathological examination of the prostate, seminal vesicles, testis, uterus and ovaries revealed no treatment-related effects.

### ***Developmental Toxicity***

No adequate data were submitted for this endpoint.

### ***Genetic Toxicity – Gene Mutation***

#### ***In vitro***

(1) *Salmonella typhimurium* strains TA92, TA98, TA100 and TA1537 were exposed to 2-nitropropane in DMSO at concentrations of 0 (control), 0.11, 0.03 or 0.1 mL/plate in the presence and absence of metabolic activation. *Salmonella* strains TA98 and TA100 were also exposed at 0.0037 mL/plate. Positive and negative controls were tested concurrently and responded appropriately. All concentrations tested were positive in strain TA100 with and without activation. All concentrations except 0.0037 mL/plate were positive in strain TA98 with and without activation (with the exception of 0.011 mL/plate with activation and 0.1 mL/plate without activation, which was toxic). Undiluted test material (0.1 mL) was positive in TA1537 with activation and was toxic in the absence of activation. 0.1 mL/plate was positive in TA92 with and without activation and 0.03 mL/plate was positive in TA92 in the presences of activation. Positive controls responded appropriately in all strains except for TA92 (in the absence or presence of activation) or in TA1537 (without activation).

**2-Nitropropane was mutagenic in this assay.**

(2) *Salmonella typhimurium* strains TA102 and TA100 were exposed to 2-nitropropane at concentrations of 0 (control), 10, 20, 40 or 80  $\mu$ moles/plate in the presence and absence of metabolic activation. Positive control response and cytotoxic concentrations were not specified.

A positive response was noted in strains TA100 and TA102 in the presence of metabolic activation at 40 and 80  $\mu$ moles/plate, respectively.

**2-Nitropropane was mutagenic in this assay.**

(3) *Salmonella typhimurium* strains TA98, TA100 and TA102 were exposed to 2-nitropropane at a concentration of 5  $\mu$ moles/plate in the absence of metabolic activation. Positive controls, negative controls and cytotoxic concentrations were not specified. Positive results were noted in *Salmonella* strain TA100 only.

**2-Nitropropane was mutagenic in this assay.**

(4) In a study conducted by NTP, *Salmonella typhimurium* strains TA1535, TA100, TA98 and TA1537 were exposed to 2-nitropropane in DMSO at 0 (control), 100, 333, 1000, 3333 or 10,000  $\mu$ g/plate in the presence and absence of metabolic activation. Positive and negative controls were tested concurrently and responded appropriately. Cytotoxicity was noted in all strains at 10,000  $\mu$ g/plate with and without activation. Positive results were noted in strains TA1535, TA100 and TA98 with and without activation.

**2-Nitropropane was mutagenic in this assay.**

(5) In a study conducted by NTP, *Salmonella typhimurium* strains TA1535, TA100, TA98 and TA1537 were exposed to 2-nitropropane in DMSO at 0 (control), 33, 100, 333, 1000 or 3333  $\mu$ g/plate in the presence and absence of metabolic activation. *Salmonella* strains TA100 and TA98 were also exposed to concentrations of 1,666, 6,666 and 10,000  $\mu$ g/plate with and without metabolic activation. Positive and negative controls were tested concurrently and responded appropriately. Cytotoxic concentrations were not specified. Positive results were noted in strains TA100 with and without activation and TA98 with activation.

**2-Nitropropane was mutagenic in this assay.**

(6) In a study conducted by NTP, *Salmonella typhimurium* strains TA1535, TA100, TA98 and TA1537 were exposed to 2-nitropropane in DMSO at 0 (control), 33, 333, 1000, 3333 or 6667  $\mu$ g/plate and in strains NR3 and NR101 at concentrations of 0, 100, 333.3, 1000, 3333.3 or 10,000  $\mu$ g/plate in the presence and absence of metabolic activation. Positive and negative controls were tested concurrently and responded appropriately. Cytotoxicity was noted in strain TA1537 at 6,666.7  $\mu$ g/plate in the absence of activation and in strains NR3 and NR101 at 10,000  $\mu$ g/plate with and without activation. Positive results were noted in strains TA1535, TA100, TA98, NR3 and NR101 with and without activation and in strain TA1537 in the presence of metabolic activation only.

**2-Nitropropane was mutagenic in this assay.**

(7) In a study conducted by NTP, *Salmonella typhimurium* strains TA1535, TA100, TA98 and TA1537 were exposed to 2-nitropropane in DMSO at 0 (control), 100, 333.3, 1000, 3333 or 9380  $\mu$ g/plate in the presence and absence of metabolic activation. Strains TA98, TA100 and TA1537 were also exposed to 2-nitropropane at 1666 and 6666  $\mu$ g/plate. Positive and negative controls were tested concurrently and responded appropriately. Cytotoxic concentrations were not specified. Positive results were noted in strains TA100, TA98 and TA1537 with and without activation.

**2-Nitropropane was mutagenic in this assay.**

(8) See human health data at: <http://webnet.oecd.org/hpv/ui/Search.aspx>

### ***Genetic Toxicity – Chromosomal Aberrations***

#### ***In vitro***

(1) In a chromosomal aberration assay conducted by NTP, CHO cells were exposed to 2-nitropropane at 0, 500, 1600 or 5000 µg/mL in the presence and absence of metabolic activation. Positive and negative controls were tested concurrently and responded appropriately.

**2-Nitropropane did not induce chromosomal aberrations in this assay.**

(2) In a sister chromatid exchange assay conducted by NTP, CHO cells were exposed to 2-nitropropane at 0, 160, 500 or 1600 µg/mL in the absence of metabolic activation and at 0, 500, 1600 or 5000 µg/mL in the presence of metabolic activation. Positive and negative controls were tested concurrently and responded appropriately.

**2-Nitropropane did not induce sister chromatid exchanges in this assay.**

#### ***In vivo***

(1) Sprague-Dawley rats (5 – 6 males/concentration) were administered 2-nitropropane via gavage at 50 or 100 mg/kg-bw and 11 additional animals were administered 300 mg/kg-bw (concentrations > 100 mg/kg-bw were given as liquid/liquid suspensions due to insolubility). Two negative control groups (6 animals/group) and one positive control group (4 animals) were tested concurrently. Six animals/group were euthanized at 24 hours and the other 6 animals/group were euthanized at 48 hours with the exception of the positive control, which was euthanized at 24 hours (there was no 300 mg/kg-bw group for the 48-hour test point). Bone marrow samples were prepared and analyzed. Only five animals in the 300 mg/kg-bw group survived for 24 hours; therefore, there were no results for the 48-hour time period for this group. In the 300 mg/kg-bw group, there was a slight increase in the frequency of micronuclei. No other effects were noted. Positive controls were tested concurrently and responded appropriately.

**2-Nitropropane did not induce micronuclei in bone marrow erythrocytes. [ see SIAP – contradiction ]**

(2) Mice (5/sex, unspecified strain) were administered 2-nitropropane at 0 (control), 200 or 300 mg/kg-bw via intraperitoneal injection and euthanized at 24 or 72 hours, at which time bone marrow samples were prepared and analyzed. Positive and negative controls were tested concurrently and responded appropriately. The highest frequency of micronucleated polychromatic erythrocytes was 0.26%. No dose- or time-dependent increases in the number of PCEs were noted and there was no increase in the micronucleus rates at 300 mg/kg-bw at 24 or 72 hours.

**2-Nitropropane did not induce micronuclei in mice.**

(3) See human health data at: <http://webnet.oecd.org/hpv/ui/Search.aspx>

**2-Nitropropane was positive for chromosomal aberrations *in vivo*.**

### ***Additional Information***

#### ***Skin Irritation***

(1) Six albino rabbits (strain and sex not specified) were administered 2-nitropropane (0.5 mL; 97.10% pure; containing 1.01% nitroethane, 2.59% 1-nitropropane and 0.023% water by weight) on shaved, intact and abraded skin under occluded conditions for 24 hours. The test site was rinsed and scored for irritation at 24, 48 and 72 hours. No treatment-related effects were noted. All erythema and edema scored at 24 and 72 hours were 0.

**2-Nitropropane was not irritating to rabbit skin.**

(2) Rabbits (strain, sex and number not specified) were administered 2-nitropropane (97.10% pure; containing 1.01% nitroethane, 2.59% 1-nitropropane and 0.023% water by weight) on shaved skin under non-occluded conditions once/day for 5 days. No signs of toxicity or irritation were observed.

**2-Nitropropane was not irritating to rabbit skin.**

#### ***Eye Irritation***

(1) 2-Nitropropane (0.1 mL) was instilled into one eye of six New Zealand White rabbits (sex not specified). Observations were made at 24, 48 and 72 hours. The untreated eyes served as controls. The maximum eye irritation score was  $1.0 \pm 1.7$  at 24 hours. At 48 hours, the mean score was  $0.3 \pm 0.8$ . The incidence of positive ocular responses in all animals was 0. Scores for conjunctival redness and chemosis, corneal opacity and effects on the iris were not provided.

**2-Nitropropane was slightly irritating to rabbit eyes.**

(2) 2-Nitropropane (0.1 mL) was instilled into one eye of six rabbits (strain and sex not specified). Observations were made at 24, 48 and 72 hours. There was no indication of ulceration or other surface lesions. Eyes of a few rabbits showed signs of moderately excessive lacrimation at the 48- and 72-hour observations.

**2-Nitropropane was slightly irritating to rabbit eyes.**

#### ***Sensitization***

Guinea pigs (10 males/group, strain not specified) were administered 2-nitropropane (0.05 mL; 97.10% pure; containing 1.01% nitroethane, 2.59% 1-nitropropane and 0.023% water by weight) in saline as a 5% solution via intradermal injection. Positive control animals were similarly injected with a 0.3% dinitro-chlorobenzene (DNCB) and saline solution (solubilized in alcohol) and negative control animals were injected with saline. Injected sites were scored 24 hours following administration and were then injected with 0.1 mL of their respective solutions at 48 hours. Injections were repeated 2 – 3 times/week until 10 injections were made. Two weeks following the last injection, animals in each group were challenged intradermally with 0.1 mL of solution at a new site. Animals from the treatment group, positive control group and negative control group were challenged with 1% test material, DNCB solution (0.03 and 0.3%) and test material and both DNCB solutions (at different sites), respectively. Animals were observed for 48 hours following the challenge injection. During the induction phase, the first 3 injections of a

5% solution of test material caused necrosis. Therefore, the last seven injections were made with a 1% solution. None of the animals that were induced with the test material reacted after challenge with the test material. In the positive control group, 8/10 animals challenged with 0.03% DNCB and all animals challenged with 0.3% DNCB had skin reactions at 24 hours. At 48 hours, all positive control animals injected with 0.3% DNCB had positive reactions. All negative control animals challenged with 0.3% DNCB had skin reaction as 24 and 48 hours, and none of the negative controls challenged with 0.03% DNCB had skin reactions.

**CASRN 79-46-9 was not a sensitizer in guinea pigs.**

### *Carcinogenicity*

(1) In the 6-month repeated-dose inhalation toxicity studies described above, rats exhibited multiple hepatocellular carcinomas. Numerous neoplastic nodules were present in the livers of all high-concentration rats at 6 months. In many cases, the normal hepatic parenchyma was destroyed. The neoplasms were composed of anaplastic hepatocytes, sometime forming broad sheets or trabeculae several cells thick. Blood-filled cysts were occasionally seen in the neoplasm. Mitotic figures were commonly observed. The carcinomas appeared to be rapidly growing. However, no metastatic hepatocellular carcinomas were seen in any of the other tissues examined. No evidence of carcinogenicity was found in rabbits that were co-exposed for 6 months to the same 2-nitropropane concentrations as rats in the same inhalation chambers.

**2-Nitropropane was carcinogenic in this study.**

(2) In the 18-month repeated-dose inhalation toxicity study described above, the total number of tumors in the unexposed group (43) exceeded the total number of tumors in the exposed group (25). The total number of animals with tumors in the control group (28) was also greater than the exposed group (23). The types of tumors observed in exposed and control females were similar. The most common benign and malignant tumors in control and exposed females were pituitary adenoma and mammary gland adenocarcinoma, respectively. The type of tumors observed in exposed and control males were different. The most common benign and malignant tumors in control males were pituitary adenoma (6) and fibrosarcoma of the skin and sutcutis (1). In exposed males, the most common benign tumor also was pituitary adenoma (1); however, the most common malignant tumor in exposed males was hepatocellular carcinoma (7).

**2-Nitropropane was not carcinogenic in this study.**

(3) In the 22-month repeated-dose inhalation toxicity study described above, there was no treatment-related effect on the incidence, distribution or total number of malignancies.

**2-Nitropropane was not carcinogenic in this study.**

(4) A retrospective mortality study was initiated to determine if there were any unusual disease mortality patterns among Louisiana workers who were born on or after the beginning of production of 2-nitropropane. The study included the 1815 employees who had worked at the plant from 1946 up until 1981. There was no unusual cancer or other disease mortality pattern among the workers before or after the beginning of 2-nitropropane production in 1955. However, since the cohort was small and the period of latency was short for most of the subjects, the study did not demonstrate that 2-nitropropane was not carcinogenic in humans.

(5) See human health data at: <http://webnet.oecd.org/hpv/ui/Search.aspx>

### ***Other***

In a dominant lethal study, male Sprague-Dawley rats (10/group) were exposed by whole-body inhalation to 2-nitropropane as a vapor at 0 (control), 25 or 200 ppm (~ 0, 0.09 or 0.7 mg/L) 7 hours/day for 5 days. Additional animals received the positive control substance via gavage for 5 consecutive days. Following treatment, two females were each introduced to one treated male. Males were separated from the females on day 12 (7 days after the last exposure). On day 22, females were euthanized and examined for pregnancy and dominant lethal effects. The mating procedure was repeated and pregnancy status was examined on each of the next 9 consecutive weeks. Effects at the highest concentration included loss of muscle tone by day 3 and decreased body weight. Pregnancy frequencies were 75 – 95 and 90 – 100% in high- and low-concentration groups, respectively, versus 80 – 100% in the control group. A small reduction in the frequencies of live implantations and late deaths were observed in high-concentration females from the second mating group only. No signs of toxicity were observed in the low-concentration group and there were no treatment-related effects on the number of corpora lutea or number of total implantations per pregnancy.

**LOAEL (maternal toxicity) = ~ 0.7 mg/L** (based on loss of muscle tone and decreased body weight)

**NOAEL (maternal toxicity) = ~ 0.09 mg/L**

**NOAEL (reproductive toxicity) = ~ 0.7 mg/L** (based on no treatment-related effects at the highest concentration tested)

**Conclusion:** The acute oral and dermal toxicity of CASRN 79-46-9 is low in rabbits. The acute inhalation toxicity of CASRN 79-46-9 is high in rats. In the 6-month repeated-dose inhalation toxicity study in rats, CASRN 79-46-9 showed increased liver weights, increased serum glutamic-pyruvic transaminase (SGPT) activity and liver histopathology and liver basophilic foci with hepatocyte hyperplasia and hyperchromatic nuclei at 0.8 mg/L/day; the NOAEC for systemic toxicity is 0.1 mg/L/day. In an 18-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed decreased body weight, increased liver weights, liver lesions at necropsy, increased SGPT, liver histopathology including focal necrosis, vascular degeneration and nodular hyperplasia and increased renal calcification at 0.4 mg/L (lowest concentration tested); the NOAEC was not established. In a 22-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed increased absolute and relative liver weight, focal vacuolization of hepatocyte cytoplasm and kidney calcification at 0.09 mg/L/day (lowest concentration tested); the NOAEC was not established. In a 6-month inhalation repeated-dose toxicity study in rabbits, CASRN 79-46-9 showed congestion and hemorrhage of the lungs with interstitial edema and necrosis in the hemorrhagic areas at 0.8 mg/L after 28-days; the NOAEC for systemic toxicity is 0.1 mg/L/day. No specific reproductive toxicity studies are available. However, in the 22-month inhalation repeated-dose toxicity study in rats, CASRN 79-46-9 showed no treatment-related effects on the reproductive organs (prostate, seminal vesicles, testis, uterus and ovaries). In the inhalation dominant lethal study in rats, CASRN 79-46-9 showed no treatment-related effects. CASRN 79-46-9 induced gene mutations in bacteria *in vitro* and chromosomal

aberrations in mammalian cells *in vivo*. CASRN 79-46-9 did not induce chromosomal aberrations or dominant lethal mutations in mammalian cells *in vitro* and *in vivo*, respectively. CASRN 79-46-9 is irritating to rabbit eyes, not irritating to rabbit skin and not a skin sensitizer in guinea pigs. CASRN 79-46-9 increased the incidence of liver and lung tumors in rats via the oral route. CASRN 79-46-9 increased the incidence of tumors in rats exposed via the inhalation route but not in rabbits.

<b>Table 3. Summary of the Screening Information Data Set as Submitted under the U.S. HPV Challenge Program – Human Health Data</b>	
<b>Endpoints</b>	<b>SPONSORED CHEMICAL 2-Nitropropane (79-46-9)</b>
<b>Acute Oral Toxicity LD<sub>50</sub> (mg/kg)</b>	<b>500 – 750</b>
<b>Acute Dermal Toxicity LD<sub>50</sub> (mg/kg)</b>	<b>&gt; 2000</b>
<b>Acute Inhalation Toxicity LC<sub>50</sub> (mg/L)</b>	<b>1.5 (male) 2.6 (female)</b>
<b>Repeated-Dose Toxicity NOAEC/LOAEC (mg/L/day)</b>	<b>(rat; 6 months) NOAEC~ 0.1 LOAEC ~ 0.8  (rabbit; 28-day/6 month) NOAEC~ 0.1/0.8 (highest concentration tested) LOAEC ~ 0.8</b>
<b>Reproductive Toxicity</b>	No effects on reproductive organs in 22-month inhalation repeated-dose study with rats.
<b>Developmental Toxicity</b>	<b>No adequate data</b>
<b>Genetic Toxicity – Gene Mutation <i>In vitro</i></b>	<b>Positive</b>
<b>Genetic Toxicity – Chromosomal Aberrations <i>In vitro</i></b>	<b>Negative</b>
<b>Genetic Toxicity – Chromosomal Aberrations <i>In vivo</i></b>	<b>Positive</b>

<b>Table 3. Summary of the Screening Information Data Set as Submitted under the U.S. HPV Challenge Program – Human Health Data</b>	
<b>Endpoints</b>	<b>SPONSORED CHEMICAL 2-Nitropropane (79-46-9)</b>
<b>Additional Information</b> <b>Skin Irritation</b> <b>Eye Irritation</b> <b>Sensitization</b> <b>Carcinogenicity</b>	 <b>Negative</b> <b>Positive</b> <b>Negative</b> <b>Positive</b>

**Bold = measured data**

#### **4. Hazard to the Environment**

A summary of aquatic toxicity data submitted for SIDS endpoints is provided in Table 4. Summary data can be gathered from: "The Dow Chemical Company. 2010. IUCLID for 2-Nitropropane: Chapter 6: Ecotoxicological Information <http://apps.echa.europa.eu/registered/registered-sub.aspx>. (Submitted to ECHA 2010)."

##### ***Acute Toxicity to Fish***

(1) Fathead minnow (*Pimephales promelas*) were exposed to CASRN 79-46-9 at unreported concentrations for 96 hours. The toxicity reported is based on the measured concentration (EHC 138).

**96-h LC<sub>50</sub> > 210 mg/L**

(2) Fathead minnow (*Pimephales promelas*) were exposed to CASRN 79-46-9 at five unspecified nominal concentrations for 96 hours under static conditions. The test substance concentrations were measured at the beginning and end of test. Five fish per two replicates for a total of 10 fish were exposed to each concentration tested. Control mortality was less than 10% at each tested concentration. Water test conditions included reconstituted water hardness of 40-48 mg/L as CaCO<sub>3</sub>, 22±1° C water temperature, and pH of 7.2-7.9.

**96-h LC<sub>50</sub> > 612.5 mg/L**

##### ***Acute Toxicity to Aquatic Invertebrates***

Water fleas (*Daphnia magna*) were exposed to CASRN 79-46-9 at measured concentrations of 5.9, 11, 21, 43, 87 and 168 mg/L under flow-through conditions. Water conditions included: water temperature of 20±1° C; dissolve oxygen of ≥ 8.4 mg/L; pH of 8.0-8.2; and water hardness of 144 mg/L as CaCO<sub>3</sub>.

**48-h EC<sub>50</sub> = 19 mg/L**

##### ***Toxicity to Aquatic Plants***

Green algae (*Pseudokirchneriella subcapitata*) were exposed to CASRN 79-46-9 at analytically geometric mean measured concentrations of 7.8, 11, 90, 379, and 887 mg/L. Sealed vessels were used to minimize volatilization of the test material. Water conditions included: water temperature of 23° C; pH of 8.0-10.8.

**72-h EC<sub>50</sub> (biomass) = 267 mg/L**

**72-h EC<sub>50</sub> (growth rate) > 887 mg/L**

**Conclusion:** The 96-h LC<sub>50</sub> value of CASRN 79-46-9 for fish is >210 mg/L. The 48-h EC<sub>50</sub> value of CASRN 79-46-9 for aquatic invertebrates is 19 mg/L. The 72-h EC<sub>50</sub> values of CASRN 79-46-9 for aquatic plants are 267 and > 887 mg/L, respectively, for biomass and growth rate.

<b>Table 4. Summary of the Screening Information Data Set as Submitted under the U.S. HPV Challenge Program –Aquatic Toxicity Data</b>	
<b>Endpoints</b>	<b>SPONSORED CHEMICAL 2-Nitropropane (79-46-9)</b>
<b>Fish 96-h LC<sub>50</sub> (mg/L)</b>	<b>&gt; 210</b>
<b>Aquatic Invertebrates 48-h EC<sub>50</sub> (mg/L)</b>	<b>19</b>
<b>Aquatic Plants 72-h EC<sub>50</sub> (mg/L) (biomass) (growth rate)</b>	<b>267 &gt; 887</b>

**Bold = experimental data** (i.e., derived from testing).

## 5. References

Environmental health criteria 138 (<http://www.inchem.org/documents/ehc/ehc/ehc138.htm>)

Haas-Jobelius, M, Coulston, F and Korte, F. (1992) Effects of short-term inhalation exposure to 1-nitropropane and 2-nitropropane on rat liver enzymes. *Ecotoxicology and Environmental Safety*, 23(3):253-259.